

Rachford (B. K.)

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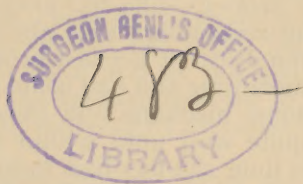
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ANÆMIA OF TUBERCULOSIS.*

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THE following case, with which I introduced my subject, is rather old to appear in pediatric literature. But it was the study of this case which led me to make the investigation of which this paper is a preliminary report, and it is therefore proper that it should serve as an introduction to a subject of vast importance to pediatricists, viz., the anæmia of tuberculosis.

CASE.†—November 4th, 1891.—Miss A., æt. twenty, came to my office for treatment. All of her family except her father had died of pulmonary tuberculosis. Her mother, her stepmother, two grown brothers and one grown sister died of this disease. Three of these deaths were within the last five years, and she was at home during the sickness of all of them. Her father and herself alone survive, and the father is at present confined

* Read before the American Pediatric Society, Boston, May 3, 1892.

† This case was one of two which I reported in the *N. Y. Medical Journal*, May, 1892. It is here reported in greater detail and for another purpose.

to his room with pulmonary tuberculosis and she nurses and cares for him.

A worse family history, with more constant exposure to the tubercular contagion is rarely seen.

Personal history.—She has always enjoyed fairly good health; she remembers that about two years ago, just following the death of her last brother, she was not quite well for a short time and had some “kernels” in her neck; she took some medicine and they disappeared and she has been quite well until within the last three months. During this time she has had more or less dyspnœa and pain in side on exercising, but she thought nothing of these symptoms and continued looking after the sick father and doing the work about the house; at the present time these symptoms have increased to such an extent that she can scarcely “keep up.”

She comes to-day complaining of great weakness, a tired feeling, considerable dyspnœa, some pain in left side, little appetite, sick stomach and great nervousness. There is no fever and no cough.

The menstrual flow during the last few months has been very slight and almost colorless. Last month she had “no show” at all.

Physical examination.—No perceptible disease of the lungs, the heart sounds normal, *lymphatic glands not noticeably enlarged*; the most pronounced and striking symptom is the extreme anæmia, as manifested in the general pallor of the skin and loss of color in the mucous membrane.

Examination of blood.—Only twenty-five per cent. of hæmaglobin and 1,680,000 red corpuscles to the cubic millimetre.

Treatment.—Three grains of reduced iron and one-fortieth grain arsenious acid three times a day.

November 18th.—Hæmaglobin thirty per cent. She thinks she is a little better; changed treatment to saccharated carbonate of iron, ten grains three times a day.

November 29th.—Hæmaglobin forty per cent. Is much better; can do her housework without much fatigue, but she is still very pale and has some dyspnœa on exercise. On the 23d she had “some show” at her menstrual time lasting a few hours.

December 1st.—Hæmaglobin fifty-two per cent. Improving in every way; eats and sleeps well; nervousness and shortness of breath are rapidly disappearing. She is getting

tired of the medicine. Changed treatment to Warner's compound chalybeate pills, two after each meal.

December 10th.—Hæmaglobin sixty-five per cent. She says she is well and that it is not necessary for her to continue taking medicine. She was persuaded to continue treatment from this time on, because she could see for herself in the hæmaglobinometer that her blood had much less color than it ought to have, and as she had noted with me from time to time the gradual increase in the color of her blood, she was the more easily induced to continue the treatment.

December 17th.—Hæmaglobin seventy per cent.

December 29th.—Hæmaglobin seventy-eight per cent. Changed treatment to three grains reduced iron, one-twentieth grain arsenious acid, three times a day.

January 18th.—Hæmaglobin ninety per cent. Last monthly time was normal.

February 1st.—Hæmaglobin ninety per cent. Stopped all medicine after nearly three months of continuous treatment.

February 17th.—Hæmaglobin ninety per cent. Corpuscles, 4,000,000 to the cubic mm. She is "perfectly well;" she "put in" a load of coal during the past week without getting tired.

March 10th.—Girl remains well. She is kept closely at home nursing her father, who is confined to bed with consumption.

March 18th.—She has not been taking medicine for about seven weeks; she says she is well. Examined her blood and was surprised to find only sixty-five per cent. of hæmaglobin and 3,200,000 red corpuscles to the cubic mm. She went two weeks over her last monthly time and it was very scanty. She protests that she is not at all sick, but I again prevailed on her to take medicine so as to keep her blood state up as near the normal as possible during the time she was nursing her father. I again gave her iron and arsenious acid.

March 30th.—Hæmaglobin ninety per cent. Feels well; discontinued medicine and ordered to report in two weeks.

In this case I would call special attention to—1st, the very bad tubercular history; 2d, the constant exposure to contagion; 3d, the "kernels" in her neck two years ago; 4th, no evidence of pulmonary or glandular tuberculosis at the present time; 5th, the state of the blood

(twenty-five per cent. of hæmaglobin); 6th, the improvement of the blood state after treatment (ninety per cent. of hæmaglobin); 7th, the rapid diminution in hæmaglobin after treatment was discontinued (to sixty-five per cent. in seven weeks); 8th, absence of symptoms when blood contained sixty-five per cent. or more of hæmaglobin; 9th, the remarkable fact that this girl has escaped pulmonary tuberculosis for so long a time.

The relationship of anæmia to tuberculosis in this case is by no means clear, and it was for the purpose of inquiring into this relationship and answering certain other questions suggested by this case that the investigation, of which I here present a preliminary report, was begun.

The table below comprehends 166 blood examinations; 164 of these were convent girls; Nos. 1 and 2 were private cases. The great majority of the girls examined were between the ages of twelve and eighteen. The convent girls were examined at the Convent of Good Shepherd in Cincinnati, Ohio, and Newport, Ky. All except thirty-six of these girls belong to the reform class; this class is made up of young girls who have gone astray and are placed in the convent for reformation. The girls of this class, therefore, do not enter the convent until they are from ten to fifteen years of age. The lives of most of these girls prior to the entering of the convent is one of wretchedness; many of them no doubt lived in badly ventilated apartments surrounded by sickness, poverty and crime. These facts are of much importance in studying the physical condition of these girls. The thirty-six referred to as not belonging to the reform class are in the "preservation class;" this class is made up of homeless girls who have been taken when quite young, some of them entering the convent at the age of three, four and five years. Most of the girls in this class have, therefore, grown up in the convent, and have had good food and proper hygienic surroundings. In the table these girls are marked with a star (*).

The following information is embraced in this table: The name, age, length of time in convent, family history

of tuberculosis, exposure to the tubercular contagion, age at first menstruation, regularity or irregularity of the menstrual function, percentage of hæmaglobin, number of corpuscles in some of the cases, and points in the personal history in each case.

The following table was arranged by Dr. Robert Carothers, who assisted me in this work.

The 166 cases recorded below were taken just as they came in the two convents; they were not selected cases. All of them considered themselves fairly well and worked every day, and none of them were under treatment at the time examined.

On close examination a few of these girls were found to have a cough, pain in the side and dyspnœa on exercise; but these symptoms were not complained of and they would not have told them unless closely questioned.

As one would expect, the family history of many of these girls is absolutely negative, but by careful inquiry and with the kind assistance of the Sisters of the Good Shepherd, I obtained a fairly good family history of many of them.

In fifty-eight cases the family history was negative; in thirty the family history was good, no tuberculosis, and in seventy-eight there was a family history of tuberculosis.

In the seventy-eight cases with tuberculous family histories the average amount of hæmaglobin was $78\frac{3}{4}$ per cent. In the fifty-eight cases with negative family histories the average amount of hæmaglobin was $85\frac{1}{2}$ per cent. In the thirty cases with non-tubercular histories the average amount of hæmaglobin was eighty-eight per cent.

These figures, so far as they go, indicate that family tuberculosis predisposes to anæmia, but they are subject to this criticism, that there may have been sufficient active tuberculosis in Class I. (with tubercular histories) to account for the ten per cent. diminution of hæmaglobin in the class. These figures are alone, therefore, of little value in solving the problem of the relationship of the

Name.	Age.	In house. Years.	Tubercular History.	Exposure to contagion.	First Menstruation.	Menstrual Function.	Per Cent. Hb.	No. Corp. per Cm.	Personal History.
1 A. L.	20		All family	Yes		Irregular	25	1,680,000	Kernels in neck 2 yrs. ago.
2 S. P.	20		Mother and aunts	"		"	30	2,250,000	Kernels in neck 2 yrs. ago, dysp., pain in side
3 R. K.	19	4	2 Bros. and 2 sisters	"	14	Missed 4 mos.	50	2,650,000	Dysp., pain in side
4 M. S.	18	12	Negative	Neg.	15	Scanty, little col.	60	2,800,000	"
5 E. M.	15	$\frac{1}{2}$	Fath., moth., 3 bros.	Yes	Never	Never	50	2,375,000	Scrofulosis, pain in side, dysp.
6 M. R.	14	$\frac{3}{8}$	Father and mother	"	13	Once in 6-7 mos.	55		Scrofulosis, pain in side, dysp.
7 A. W.	17	$\frac{1}{4}$	Half-bro. and uncle	"	14	Regular	45		Scrofulosis, pain in side, dysp.
8 A. B.	16	$\frac{1}{8}$	Mother and brother	"	14	Irregular	45		Scrofulosis
9 L. K.	17	$\frac{1}{2}$	Sister	Neg.	14	{ Metrorrhagia Menorrhagia	35		Small and pale
10 A. K.	14	2	Mother	No	Never	Never	60		
11 S. F.	15	3	Mother and sister	Yes	13	Irregular	65	2,533,000	
12 E. K.	18	$\frac{7}{8}$	2 Brothers	"	15	"	60		
13 M. W.	16	$\frac{1}{8}$	Mother and sister	"	13	"	65	2,543,000	Dyspnœa
14 R. K.	23	$\frac{3}{8}$	2 Brothers	No	14	"	63	4,500,000	"
15 B. G.	18	1	Mother	Yes	15	Slight and pale	60		Had kernels in neck 2 yrs. ago, pain in side, dysp.
16 M. H.*	17	2	"	No	16	Irregular	65		Dysp., pain in side
17 M. S.*	14	8	Negative	Neg.	13	Regular	63		Scrofulosis
18 C. L.	16	16	"	"	Neg.	Negative	65		Small and delicate
19 I. R.	14	2	Mother	Yes	14		65		Dysp., pain in side
20 M. M.*	14	3	"	"	Never		65		Diarrhœa 2 yrs.

21 M. M. *	10	1	Sister	Yes	10	Slightly once	65	Dyspnœa
22 A. M.	28	10	Negative	Neg.	18	Irregular	65	Slight cough
23 C. F.	17	2	Good	No	15	"	60	Cough and dysp.
24 A. L.	16	$\frac{1}{2}$	Negative	Neg.	19	Negative	67	Scrofulosis
25 A. N.	31	10	"	Neg.	17	Regular	75	Had hæmorrhage
26 M. A.	19	4	Mother and sister	Yes	15	"	75	Pain in side
27 A. B.	23	2 $\frac{1}{2}$	Mother and brother	"	16	{ Metrorrhagia	3,460,000	
28 M. M.	27	3	3 Aunts and brother	No	14	{ Menorrhagia	3,600,000	
29 R. T.	20	6	Mother and sister	Yes	16	Irregular	2,620,000	
30 M. R.	19	1	Negative	Neg.	12	"	3,012,000	
31 M. E.	17	10	Good	No	13	Regular	70	Pain in chest, dysp
32 B. C. *	16	8	Mother	Yes	14	Irregular	75	Dyspnœa
33 A. L. *	17	$\frac{3}{4}$	Sister	"	13	"	70	
34 K. M. *	16	10	Brother and sister	No	12	Regular	75	
35 P. P. *	13	14	Sister	Yes	15	"	70	Been taking iron,
36 A. B. *	16	4	Mother	No	15	Irregular	75	scrofulosis
37 N. T. *	14	4	Good	No	Never	Regular	70	
38 B. E.	22	6 $\frac{1}{2}$	Moth., bro., sister	Yes	17	Irregular	75	Dyspnœa
39 M. N.	18	8	Negative	Neg.	13	"	70	"
40 C. F.	17	4	Sister	Yes	14	Regular	75	Cough
41 M. B.	21	12	Father	"	11	"	75	Scrofulosis
42 K. D.	32	11	Mother	No?	15	"	70	
43 A. McM.	20	1 $\frac{2}{3}$	Fath., sister, uncles	Yes	14	Irregular	75	
44 K. H.	37	$\frac{1}{2}$	Father	Neg.	Never	Regular	70	
45 C. W.	14	1 $\frac{1}{2}$	Negative	Yes	15	"	75	
46 L. R.	14	4	Father and mother	No	14	Irregular	70	
47 A. B.	21	5	Good	Neg.	16	Regular	75	Scrofulosis
48 M. B.	20	3	Negative	Yes	15	"	70	
49 M. D.	29	6	Mother and brother	"	16	Irregular	77	
50 M. B.	25	1 $\frac{1}{2}$	Mother	Neg.	Neg.	Regular	75	
51 L. P.	16		Negative	Neg.				

Name.	Age.	In house Years.	Tubercular History.	Exposure to contagion.	First Menstruation.	Menstrual Function.	Per Cent. Hb.	No. Corp. per Cm.	Personal History.
52 M. C.	38	18	Negative	Neg.	14	Regular	75		
53 M. E.	18	1 ¹ / ₂	Mother and sister	Yes	14	"	80		
54 U. B.	11	11	Father	No	12	"	80		
55 E. M.	21	3	Mother	Yes	13	Metrorrhagia	85		
56 K. K.*	17	5	Father	"	15	Irregular	80		
57 M. D.*	16	4	Mother and brother	"	14	Regular	80		Pain in side, dysp.
58 L. P.*	16	11	Sister	No	15	Irregular	85		"
59 M. H.	16	11	"	"	14	"	85		
60 C. S.	30	6	Negative	Neg.	14	Regular	85		
61 G. P.	22	6	"	"	16	Irregular	80		Insane 11 yrs. ago
62 J. I.	17	12	Good	No	14	Regular	80		
63 C. B.	21	6	Negative	Neg.	15	Irregular	80		
64 L. R.*	16	5	"	"	14	Regular	85		
65 K. P.*	15	6	"	"	13	"	80		Exophthalmic goitre
66 A. R.	14		"	"		Irregular	85		
67 M. D.*	16	4	"	"		Regular	85		
68 M. T.*	14	4	Good	No	13	"	88		
69 N. T.	14	4	"	"	Never		85		
70 J. U.	21	3	Mother and sister	"	19	Irregular	85		
71 M. B.	16	1 ¹ / ₂	Sister	Yes	12	"	85		
72 R. D.	18	2	Good	No	13	Regular	80		Pain in side, dysp.
73 M. A.	21	2	"	"	16	"	85		
74 R. L.	18	5	"	"	12	"	85		
75 T. W.	16	1 ¹ / ₂	"	"	Never		80		Dyspncea
76 M. I.	19	2	Mother	Yes	12	Regular	80		
77 T. B.	16	4	Father and brother	"	13	"	80		
78 M. R.	16	3	Good	No	14	"	80		
79 A. H.	16	1	Negative	Neg.	12	Irregular	85		

81 M. K.	25	12	All family	Yes	14	Irregular	85	Cough
82 F. T.	18	1 $\frac{1}{2}$	Father	"	14	Regular	80	Slight cough
83 L. L.	30	16	Good	No	14	"	80	
84 M. B.	12	9	"	"	Never	"	85	Well
85 M. W.	14	$\frac{2}{3}$	Mother and brother	Yes	"	"	77	
86 E. F.	14	$\frac{1}{4}$	Negative	Neg.	"	"	80	
87 A. F.	21	4	Good	No	12 $\frac{1}{2}$	Regular	80	
88 M. M.	17	4	"	"	13	Irregular	83	
89 M. D.	30	15 $\frac{1}{2}$	Negative	Neg.	16 $\frac{1}{2}$	Regular	85	
90 E. G.	32	18	"	"	18	"	80	
91 K. H.	17	1 $\frac{1}{2}$	Bro., sis., moth., aunt	Yes	"	Irregular	80	Pain in side
92 E. B.	15	3	Negative	Neg.	"	Regular	85	Well
93 M. T.	16	1 $\frac{1}{6}$	Mother	Yes	15	"	100	Dyspnœa
94 S. J.	25	2 $\frac{1}{2}$	Uncle	No	10	"	100	Pain in side
95 J. S.	15	$\frac{1}{2}$	Mother	Infancy,	Never	"	95	Pain in side, dysp.
96 J. L.	30	4	Mother and brother	Yes	12	Slight, dark	100	Hæmorrhage
97 J. S.	13	$\frac{1}{2}$	Mother	Infancy,	Never	"	95	Dyspnœa
98 E. O.	28	5	Uncles and aunts	No	16	Regular up to 2	90	3 hæmorrhages from
99 B. K.	26	6	Moth., sis., bro.	Yes	13	months ago	95	stomach
100 J. M.	16	14	Mother	"	13	Metrorrhagia	100	Cough, dysp., pain
101 C. T.	16	2 $\frac{1}{2}$	Mother and sister	"	13	Regular	90	Well
102 M. A.	14	1 $\frac{1}{2}$	Mother	"	13	"	100	"
103 L. M.	14	$\frac{1}{4}$	Uncles	No	12 $\frac{1}{2}$	"	90	
104 L. M.	21	$\frac{1}{4}$	Mother	Yes	16	"	90	
105 K. Z.	17	$\frac{1}{2}$	Mother and uncle	"	15	Irregular	100	
106 M. T.	24	6	Mother	"	"	Regular	100	
107 E. B.	23	9	Mother and brother	No	"	"	100	
108 J. L.*	17	8	Sister	Infancy,	"	"	100	
				Yes				

Name.	Age.	In house. Years.	Tubercular History.	Exposure to contagion.	First Menstruation.	Menstrual Function.	Per Cent. Hb.	No. Corp. per Cm.	Personal History.
109 M. O.*	17	1 ¹	Sister	No	13	Regular	90		
110 N. K.*	15	4	Father and sister	Yes	13	Irregular	100		Dysp., pain in side
111 M. D.*	16	8	Mother and brother	No	14	Regular	90		
112 K. C.	25	15	Good	"	16	Metrorrhagia	100		
113 H. G.	22	5	Negative	Neg.	16	Regular	100		
114 M.	35	2	"	"	14	"	100		
115 K. S.	25	6	"	"	16	"	95		Dyspnœa
116 F. M.	18	1 ¹	Good	No	16	Irregular	100		
117 J. D.	19	1	Negative	Neg.	14	Regular	100		
118 A. H.	17	3	Good	No	13	"	100		
119 L. O.	19	2	Negative	Neg.	16	"	95		
120 F. W.	18	10	"	"	15	"	90		
121 K. H.	17	4 ¹	Good	No	16	"	90		
122 C. R.	17	12	Negative	Neg.	13	"	90		
123 K. G.	17	12	"	"	13	"	90		
124 M. M.*	16	3	Good	No	13	"	100		
125 M. H.*	16	14	Negative	Neg.	13	"	90		
126 A. K.*	17	3	"	"	16	"	90		
127 M. D.*	16	8	"	"	15	"	90		
128 M. G.*	21	3	"	"	17	"	90		
129 T. M.*	17	1	"	"	16	"	100		
130 A. P.*	15	10	"	"	12	"	90		
131 M. R.*	13	5	"	"	Never		100		
132 M. E.*	13	2	"	"	"		95		
133 W. R.	13	5	"	"	"		90		
134 W. E.	14	2 ³	Good	No	"		90		Looks delicate
135 G. C.	12	2	Negative	Neg.	"		95		
136 A. H.	16	3	"	"	9	Regular	95		

137 M. L.	16	2	Negative	Neg.	15	Regular	100	
138 J. C.	19	1	"	"	16	"	100	Old scrofulosis
139 L. S.	21	3	Brother	Yes	20	Metrorrhagia	90	
140 M. C.	16	3	Good	No	13	Regular	100	Pain
141 A. S.	17	3	Negative	Neg.	14	Irregular	100	
142 F. E.	15	1	Aunts	Yes	12	Regular	100	
143 K. W.	23	10	Negative	Neg.	13	"	90	
144 E. M.	16	7	Good	Neg.	14	Irregular	100	
145 K. P.	21	12	Negative	Neg.	16	"	93	
146 N. K.	15	11	"	No	13	Regular	100	
147 C. D.	21	8	Good	"	18	"	100	
148 F. S.	16	14	Brother	Yes	14	"	100	Pain in side, dysp.
149 A. S.	16	14	Good	No	14	Irregular	95	"
150 M. G.	16	3	Negative	Neg.	14	Regular	95	"
151 M. J.	16	3	Father and sister	Yes	14	Irregular	100	
152 H. L.	22	5	Mother	Yes	14	Regular	100	
153 A. K.	23	8	Good	No	15	"	100	
154 A. S.	31	7	"	"	15	Irregular	100	
155 A. R.	21	13	"	"	15	"	95	
156 A. B.	16	14	"	"	14	Regular	95	
157 M. O.	39	11	Negative	Neg.	12	Negative	95	
158 L. B.	13	14	"	"	14	Regular	95	
159 A. T.	15	13	"	"	12	Negative	95	
160 E. G.	15	13	"	"	14	Regular	90	Scrofula?
161 A. C.	37	1	"	"	Never	Irregular	95	
162 L. E.	17	4	Father	Neg.	14	Negative	90	
163 T. W.	25	4	Sister	Yes	18	Regular	95	
164 M. M.	30	10	Negative	No	18	"	90	
165 A. L.	44	24	"	Neg.	20	"	90	
166 L. D.	30	3	Mother.	Yes	Neg.	"	90	

anæmia to the tuberculosis. But if we exclude all the cases of tuberculosis and scrofula found in the tables, we find that the above percentages are not materially changed—we then have sixty-nine cases with tubercular family histories, but having no apparent tuberculosis with an average of $80\frac{1}{2}$ per cent. of hæmaglobin. This percentage, when compared with the eighty-eight per cent. in the thirty cases with non-tubercular histories, affirms the conclusion arrived at above that there is a relationship between tuberculosis and anæmia, apart from the anæmia that is produced by *apparent* active tubercular disease.

By further study of the table we learn that fifty-two of the 166 girls examined were decidedly anæmic, having seventy-five per cent. less of hæmaglobin. Of the fifty-two anæmic cases only four, or about seven-and-a-half per cent., have good family histories, no tuberculosis; twelve, or about twenty-three per cent., had negative family histories, and thirty-six, or about seventy per cent., had tubercular family histories. Among the remaining 114 cases with more than seventy per cent. of hæmaglobin there were twenty-six, or twenty-three per cent., with non-tubercular histories; forty-five, or about thirty-nine per cent., with negative histories, and forty-three, or about thirty-seven-and-a-half per cent. with tubercular histories. Here we have two sets of figures worth comparative study, which we may for convenience arrange in the following manner:

	No tubercular family history.	Tubercular family history.	Negative family history.
52 cases, anæmia with less than 75% Hb.,	7½%	70%	23%
114 cases, non-anæmic, with more than 75% Hb. . . .	23 %	37%	39%

The contrast between these two sets of figures is striking and the percentages are made from a sufficient number of cases to make their study valuable. These figures again show that there is a relationship between family tuberculosis and anæmia; a very small percentage of

anæmia cases have a family history free from tuberculosis, and a comparatively large percentage of the non-anæmic cases have good family histories. We conclude, therefore, that girls from tubercular stock are much more likely to be anæmic than girls from non-tubercular stock, or in other words, that family tuberculosis is one of the great sources of anæmia.

The following diagram is made from the same group of cases, except that all the cases of apparent pulmonary and glandular tuberculosis have been omitted. It will be seen that this omission does not materially alter the figures given above.

	No tubercular family history.	Tubercular family history.	Negative family history.
37 anæmic cases, 75% or less of Hb., with no apparent tu- berculosis,	74 $\frac{3}{4}$ %	72%	20 $\frac{1}{2}$ %
100 non-anæmic cases. More than 75% of Hb., with no apparent tuberculosis . .	25%	40%	45%

The contrast in these two sets of figures is just as striking as in those given above, and these clearly prove that girls from tubercular stock, even though they be *apparently* free from tuberculosis, are very much more likely—in the ratio of seventy-two to seven-and-three quarters per cent.—to be anæmic than girls with non-tubercular family histories. This gives great importance to the tubercular or non-tubercular family histories in anæmic cases, since family tuberculosis is here shown to be one of the important causes of anæmia.

Let us now give attention to the cases of tuberculosis found in the tables. There are eight cases of glandular tuberculosis with no pulmonary disease (Nos. 7, 8, 9, 17, 25, 36, 42, 48). Case 34 is excluded because she had been taking iron for some time. The average amount of hæmaglobin in these cases was fifty-seven per cent.

There are ten cases of pulmonary tuberculosis with evidence of scrofula (Nos. 22, 24, 26, 28, 41, 81, 82, 96, 98, 99) and the average amount of hæmaglobin in these cases was eighty per cent.

Fifty-seven per cent. of hæmaglobin in cases of scrofulosis and eighty per cent. in the cases of pulmonary tuberculosis make indeed a striking contrast and these figures seem to show that it is rather tubercular disease of the lymphatics than tubercular disease of the lungs that produces the most pronounced anæmia.

The cases of scrofulosis included in these tables were by no means aggravated ones. They did not complain of being sick and were not under treatment at the time examined. The diagnosis of scrofula was made by the previous history, old scrofulous scars and enlarged glands.

The cases of pulmonary tuberculosis were also in a semi-latent stage. They complained of pain in the side and dyspnœa on exercise, all of them had a slight cough and four of them had had hæmorrhages. These symptoms and the physical signs sufficient to make a diagnosis.

Three of the girls of this class, within a month after the examination, developed a sharp attack of pulmonary tuberculosis following the influenza. The cases in the two groups are, for the reasons given, fit cases for comparison. Advanced and active scrofulosis is not here compared with mild and latent pulmonary tuberculosis; neither is latent scrofulosis compared with a very advanced and active pulmonary tuberculosis.

But if there is a difference in degree in these two groups, I think that so far as external symptoms and signs go the pulmonary cases are the more pronounced. But from a critical study of these cases which are not selected, but taken as they come from 166 examinations, I see no reason to doubt the correctness of the above figures. I think, therefore, that the table presented, so far as it goes, seems clearly to show that tubercular disease of the lymphatic tissues causes a great reduction in the amount of hæmaglobin in the blood, and that beginning tuberculosis of the lungs may cause very little if any reduction at all.

Case No. 19, with history of previous hæmorrhage, the amount of hæmaglobin was normal.

While it is not the purpose of this paper to inquire into all the causes of anæmia, it must be noted that in the group of cases presented, family tuberculosis so overshadows all other causes that their consideration becomes a matter of secondary importance. If we study the influence of lack of food and bad hygiene in producing anæmia in these girls before they entered the convent, we find that those that suffered most from these causes before entering the convents, and were the slowest to recover from the effect of these influences after entering the convent, were the girls with tubercular family histories, I am led by this investigation, therefore, to believe that while lack of food and bad hygiene are undoubtedly direct causes of anæmia, it is probable that the most deleterious influence of these factors is exerted in an indirect way by predisposing to and prolonging attacks of tuberculosis.‡ This is more evident if we note the fact that anæmic girls with non-tubercular histories ordinarily recover rapidly after entering the convent. In this connection we may call attention to the fact that of the non-tubercular cases with good family histories the lowest percentage of hæmaglobin was seventy per cent. and the next lowest seventy-five per cent.

Chronic diarrhœa, which is spoken of as one of the causes of anæmia, existed in only one case (No. 20), but this case also had a bad tubercular family history. If there be other factors in producing the anæmia in the group of cases I have here reported I have failed to discover them. Clearly then, we may conclude that the above table establishes a most important relationship between anæmia and family tuberculosis. But we have yet to answer the most important query. How is the anæmia produced in those cases that have no apparent tubercular disease! Is the anæmia a pre-tubercular con-

‡ It may be here noted that tuberculosis is probably a much more important factor in producing anæmia among convent girls than it is in the cases one sees in private practice, because of the fact that most convents are notoriously tubercular communities; but this would not detract from the relative value of the figures given.

dition the result of some mysterious inheritance, or is it due to a secret or hidden tuberculosis of the deep lymphatics or other blood-forming organs?

We may now intelligently answer this question in the light of the two propositions heretofore established, viz.: 1st. Family tuberculosis is one of the great sources of anæmia. 2d. Tubercular lymphatic disease causes profound anæmia, while beginning pulmonary tuberculosis may not produce anæmia.

In considering these propositions one is led to the opinion that the anæmia in apparently non-tubercular girls coming from tubercular stock is, when not readily explainable by other causes, very probably due to a *deep-seated* and *hidden* glandular tuberculosis.

Further light may be thrown on this question by studying in the table the cases of anæmia (below seventy per cent.) that have tubercular family histories but are themselves apparently free from active tuberculosis; there are eighteen such cases and all of them except four were exposed to the contagion of tuberculosis by living in a house with a person sick of this disease. In the four not exposed§ the average percentage of hæmaglobin was 64.5 per cent. In the fourteen exposed the average percentage of hæmaglobin was fifty-seven per cent. These averages, together with the fact that all the cases (5 in number) having fifty-five per cent. and less of hæmaglobin are in the exposed class seem to indicate that exposure to tubercular contagion, as well as the tuberculous family history, plays a rôle in producing the anæmia. This is strongly confirmatory of the opinion expressed above that anæmia is due to a deep-seated disease of the blood-forming organs.

Another very important point bearing on this question is that some of these apparently non-tubercular girls give on close questioning a history of having had, several years ago, "some kernels in the neck." In such a case, with a history of previous external lymphatic enlarge-

§ By "not exposed" is meant that they did not live for any length of time in the room of one sick of tuberculosis.

ment, it requires no stretch of the imagination to believe that the anæmia is due to some hidden tubercular process. The case reported at the beginning of this paper is an example of this kind. This girl had the worst possible family history and she had been constantly exposed to the contagion of tuberculosis for years. Notwithstanding these facts, when she presented herself to me for treatment there was no evidence of tubercular disease other than the pronounced anæmia (only twenty-five per cent. of hæmaglobin). But this girl two years before "had kernels" in her neck caused no doubt by tuberculosis of the lymphatic glands, and there is now no doubt in my mind, for the reasons given in this paper, but that her present anæmic condition is due to a tuberculous disease of the deep lymphatics. This view of the case also explains why there is so great a falling off in the amount of hæmaglobin in her blood, when the iron is discontinued.

From all the arguments presented in this paper, I think I am justified in the conclusion that *pronounced anæmia, without apparent cause, is strongly suggestive of concealed tuberculosis.*

There is another question of great importance which may here be inquired into. Why has this girl, whose history I have recorded, escaped pulmonary tuberculosis? Bearing on this question, Hayem says: "The chlorotic ground," in some instances, "seems to be unfavorable to the development of the tubercle bacillus." He here uses the term "chlorotic" as synonymous with the term "chloro-anæmic," which he uses to describe the anæmic condition associated with tuberculosis. In some instances he believes that the anæmia occurring in those of tubercular stock not only is not caused by tuberculosis but actually protects against this disease by furnishing "ground unfavorable to the development of the tubercle bacilli." In other instances he believes the anæmia is a pre-tubercular condition which, after the onset of the disease, either may or may not increase in severity with the progress of the disease. In still other instances he

looks upon the anæmia as a symptom of active pulmonary or other tuberculosis which progresses with the disease. These various relations between anæmia and tuberculosis, together with the various forms of tubercular chloro-anæmia (simple anæmia, chlorosis and pernicious anæmia) which Hayem so indefinitely describes, leaves one in as much doubt as ever as to the value and meaning of anæmia in the study of tuberculosis. But it was not to offer a criticism on the work of Hayem that the above references are made, but rather to confirm in a modified form, his observation that "chloro-anæmia" in some instances "seems to protect against tuberculosis." I also have observed that some of these anæmic girls with tubercular family histories, even though they may be repeatedly exposed to tubercular contagion, escape active pulmonary tuberculosis for a surprisingly long time. The case reported is a striking example. But in the light of the preceding argument I believe that it is not the anæmia, as Hayem supposes, that gives the protection, but it is the cause of the anæmia, viz., the deep-seated and glandular tuberculosis or hidden scrofula.

In this connection may be mentioned an oft-repeated clinical observation that children who have recovered from an attack of scrofulosis seem to have a partial immunity against pulmonary tuberculosis and I may add, by way of parenthesis, that the conferring of immunity from pulmonary tuberculosis by an attack of scrofulosis is not inconsistent with the most rational theories|| of immunity as they are taught to-day.

The above explanation is given because it offers a satisfactory explanation of Hayem's apparently inconsistent observation, that in certain cases the anæmia protects against tuberculosis; for surely it is difficult to believe that the anæmia itself could in any way protect against the tuberculosis, but it is not difficult to understand that the deep-seated glandular tuberculosis which is the cause

|| See my paper "The Mechanism of Immunity," *Philadelphia Medical News*, 1892.

of the anæmia may, when recovered from, offer some protection against pulmonary tuberculosis.

In this paper the term anæmia has been used in a general sense and refers only to the diminution in the amount of hæmaglobin in the blood and has no reference to the number of corpuscles. Although my observations on the classification of the anæmia of tuberculosis are not yet ready for publication, they lead me to believe that the typical anæmia of tuberculosis approaches more nearly simple anæmia than chlorosis, while in many instances the reduction of corpuscles does not correspond to the reduction in hæmaglobin, often being less or rarely greater; yet this difference is not so great as to approach either the chlorotic type on the one hand or pernicious anæmia on the other.

Cases of chlorosis, however, do occur and they are as a rule of more than ordinary interest and are very suggestive of the importance of this field of work; for example, one case of simple anæmia was treated with iron until the amount of hæmaglobin had reached seventy-five per cent. Under the iron the corpuscles had increased proportionately, the iron was discontinued and arsenic was given for three weeks. At the end of this time the number of corpuscles was 4,500,000 to cm. and the amount of hæmaglobin had remained the same, seventy-five per cent. This case had under arsenic passed from simple anæmia to one of chlorosis. Another case¶ in which menorrhagia was a prominent symptom was decidedly chlorotic. Another very instructive case¶ of chlorosis in a colored child nine years of age was one of hip-joint disease, with extensive lymphatic involvement. The long-continued suppuration (three years) in this case had been followed by amyloid disease of the liver and kidneys. The liver in this case is enormously enlarged, occupying almost the entire abdomen. The possible connection in this case between the destruction of the liver function and the chlorotic condition of the blood

¶ These cases are not included in the tables. They belong to another group of cases examined later.

SUPPLEMENTAL TABLE.

Name.	Age.	In house.	Tubercular History.	Exposure to contagion.	First Menses	Menstrual Function.	Per Cent. Hb.	No. of Corpuscles.	Personal History.
1 J. W.	19	6	Mother	Yes	15	{ Metrorrhagia Menorrhagia	30	3,730,000	Pale
2 M. S.	18	6	"	"	15	Regular	65		Well
3 A. B.	11	6	Negative	Neg.	Never		65		Lymph. enlarged
4 B. H.	7	1 ¹	Mother	Yes	"		65		"
5 K. E.	17	10 ¹	Negative	Neg.	15	Regular	70		Well
6 M. G.	14	8 ¹	Sister, mother, father.	Yes	13	"	70	2,200,000	Glands enlarged
7 M. W.	17	8	Father and mother	"	13	Irregular	70		Pale, heart disease, valvular
8 E. E.	14	9	Negative	Neg.	13	Regular	70		Glands enlarged, goitre
9 M. H.	14	8	Mother	Yes	13	"	70		Glands enlarged
10 E. G.	14	6	"	"	Never		70		No lymph. enlarg.
11 L. T.	12	2	Father	"	"		70		Phthisis
12 C. H.	14	1 ¹	Mother	"	"		70		No lymph. enlarg.
13 R. N.	13	4	Negative	Neg.	"		70		Lymph. enlarged
14 A. H.	11	1 ¹	Mother	Yes	"		70		No lymph. enlarg.
15 K. M.	11	2	Mother, 2 brothers	"	"		75		Decided lymph enlargement
16 R. O'C.	7	4	Father	No	"		75		Decided lymph enlargement
17 M. W.	14	6	Mother	Yes	"		75	4,400,000	Pain in side, dysp.
18 K. B.	17	3 ¹	"	"	15	Regular	75	3,360,000	Scrofula
19 M. G.	18	6	Negative	Neg.	14	"	80		Well

20 M. R.	17	7	Negative	Neg.	14	Regular		80	Well
21 A. M.	16	7	"	"	15	Irregular		80	Pain in side
22 M. F.	16	3½	Father	Yes	16	Regular		80	Phthisis
23 B. T.	14	3	Brother	"	Never			80	Well
24 E. D.	16	2	Good	No	"			80	"
25 N. C.	13	½	Negative	Neg.	"			80	Phthisis
26 M. M.	13	2	"	"	"			80	Lymph. enlarged
27 K. M.	14	10	Father and mother	Yes	"			80	"
28 T. M.	12	10	"	"	"			80	Phthisis
29 A. H.	12	2½	Negative	Neg.	"			80	Well
30 E. H.	16	1½	"	"	"			85	"
31 L. S.	16	7	Father and mother	No	14	Regular		85	Lymph. enlarged
32 M. M.	13	2	Mother, 2 brothers	Yes	13	"		85	Phthisis
33 A. O'C.	13	4	Father	"	Never			85	Well
34 N. F.	13	6	Good	No	13	"		88	"
35 K. K.	18	8	"	"	14	"		100	"
36 J. P.	17	6	"	"	15	"		90	"
37 M. F.	16	8	"	"	13	"		90	Phthisis
38 M. C.	17	2	Brother	Neg.	16	"		90	Well
39 M. L.	17	4	Father and mother	Yes	16	"		100	"
40 M. M'C.	14	1	Negative	Neg.	12	"		90	"
41 M. B.	15	5	"	"	14	"		95	"
42 M. S.	16	5	Mother	Yes	14	Negative		87	"
43 A. O.	14	2	Good	No	13	Regular		90	Phthisis
44 J. F.	18	2	Father	Yes	13	"		90	Well
45 B. M'C.	16	8	Good	No	14	"		95	"
46 C. W.	14	6	"	"	13	"		90	"
47 J. W.	14	2	Negative	Neg.	Never			100	"
48 N. W.	15	10	"	"	14	Irregular		95	"
49 A. R.	14	2	"	"	Never			90	"
50 K. M'C.	15	9	Good	No	14	Regular		100	"
51 M. H.	14	2	"	"	14	"		100	"
52 M. W.	13	9	Negative	Neg.	Never			90	"
53 E. S.	11	2	Good	No	"			95	"

offers a tempting field for speculation. These interesting chlorotic cases are, as I have said, the exception and not the rule, and simple anæmia is the characteristic anæmia of tuberculosis.

As confirming the conclusions heretofore arrived at, the following points taken from the following table are worthy of note :

25	cases with tubercular family histories have an average of	75 $\frac{3}{4}$	Hb.
17	" " negative " " " "	88 $\frac{1}{4}$	"
11	" " good " " " "	92 $\frac{1}{2}$	"

Excluding the cases of *apparent* pulmonary and glandular tuberculosis we have

11	cases with tubercular family histories have an average of	72	% Hb.
12	" " negative " " " "	86 $\frac{1}{2}$	% "
11	" " good " " " "	92 $\frac{1}{2}$	% "

It is here worthy of note that in none of the children with good family histories was there any evidence of tuberculosis.

The following table shows the relationship of the anæmia to the tubercular or non-tubercular history of these girls. The eighteen cases having less than seventy-five per cent. of hæmaglobin are classed as anæmic; the thirty-five cases having more than seventy-five per cent. of hæmaglobin are classed as non-anæmic.

	No tubercular family history.	Tubercular family history.	Negative family history.
18 anæmic cases, . . .	None	14 cases, or 77 $\frac{2}{3}$ %	4 cases, or 22 %
35 non-anæmic cases, . . .	11 cases, or 31 $\frac{1}{2}$ %	11 cases, or 31 $\frac{1}{2}$ %	13 cases, or 37 $\frac{1}{7}$ %

The examinations embraced in the supplemental table on pages 170-171, were made too late to be included in the table upon which this paper was based. An examination of this supplemental table will confirm and strengthen the conclusions arrived at above. These girls belong to the preservation class of the convent of the Good Shepherd, located at Carthage, Ohio.

The following table shows the relationship of the anæmia to the tubercular family history after all cases of apparent tuberculosis have been excluded.

	No tubercular family history.	Tubercular family history.	Negative family history.
8 anæmic cases, with no ap- parent tuberculosis, . . .	None	7 cases, or 87½ %	1 case, or 12½ %
26 non-anæmic cases, with no apparent tuberculosis, . .	11 cases, or 42¾ %	5 cases, or 19¼ %	10 cases, or 38¾ %

This supplemental table also contains thirteen cases of evident tubercular disease of the lymphatics, with an average of seventy-three per cent. of hæmaglobin, and six cases of evident tubercular disease of the lungs, with 84½ per cent. of hæmaglobin.

These percentages confirm the conclusion heretofore arrived at, that it is tubercular disease of the lymphatics, rather than tubercular disease of the lungs, that produces the most pronounced anæmia. In short, we may say that a critical study of the second table confirms in every particular the conclusion drawn from a study of the first table and strengthens the deduction that *pronounced anæmia without apparent cause is strongly suggestive of concealed tuberculosis.*

This observation may throw some light on many of the obscure cases of anæmia and chlorosis that are constantly coming under observation.

DISCUSSION.

Dr. FISCHER.—I would like to state what I said in connection with the paper of Dr. Caillé. He said amongst other things that he had used ozone in the treatment of tuberculosis with no benefit whatsoever, and had had benefit in connection with chlorosis and anæmia and also in pertussis. I asked Dr. Caillé whether he had examined the secretions or excretions of patients afflicted with anæmia or chlorosis for tubercle bacilli. He said he had not. Now I am confident that, as Dr. Rachford has just said, cases of anæmia and chlorosis with nothing else

but anæmia are very often cases of true tuberculosis, because I have examined both secretions and excretions. Especially have I seen one case of a young girl with an obstinate form of chlorosis, who suffered from a severe form of diarrhœa and wherein from the discharges of the bowel I could get distinct tubercle bacilli. She had no cough whatsoever and this case was attended with anæmia for almost two years until these bacilli were found. It was then proven that she had been in a family as nurse where a young girl had died from tuberculosis. I believe this person was infected and had no other symptoms except the diarrhœa.

Dr. OSLER.—In how many cases did he find a normal percentage of hæmaglobin in the whole group examined?

Dr. RACHFORD.—I really can't tell that, but I found a great proportion of the cases above eighty-five per cent.

Dr. OSLER.—Is it not exceptional to get in young women a normal percentage of hæmaglobin?

Dr. RACHFORD.—Yes.

Dr. OSLER.—I would like to express my appreciation of the value of this paper. It seems an admirable piece of clinical observation and I think the doctor's conclusions are those which have already been arrived at, that the anæmia of tuberculosis is very commonly a simple anæmia, not chlorotic anæmia, though there are some instances undoubtedly in the early stages of tuberculosis in which the condition is what the French writers describe by the term chloro-anæmia.

Dr. ROTCH.—I want to express my appreciation of the very large amount of work that must have been done to prepare a paper of that kind.

Dr. RACHFORD.—I would say that I have besides this, and would have incorporated in this paper, quite a group of younger cases had the time permitted. I have made probably one hundred blood examinations in a convent back of Cincinnati. ~~This is not incorporated in this group and they are very much younger children, from six to ten years of age. I think nearly all of these cases substantiate the same thing as those in this paper.~~

